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THE CLINICAL RECOGNITION OF CORONARY ARTERY INSUFFICIENCY

The term "coronary artery insufficiency" or the shorter term "coronary insufficiency" has become the most widely acceptable functional description for syndromes resulting from an imbalance between coronary arterial blood supply and myocardial requirements. Another explanation has been offered for anginal pain, namely, that it arises from stimuli originating in the walls of the coronary arteries themselves, rather than from the myocardium. At present, this seems a less likely mechanism than anoxia.

Anoxia of the heart muscle may result from conditions not related to primary atherosclerosis of the coronary tree, such as obstruction to the coronary ostia, high degrees of anemia, increased metabolic demands, as in severe exertion (even in youth), or thyrotoxicosis. Such conditions are usually obvious and demonstrable. They are not conventional parts of the pattern of coronary arterial disease which carries with it the threat of sudden death, except as they may complicate coronary atheroma or fibrosis.

The term "coronary insufficiency," as used in this paper, implies degenerative arterial disease. In its incipiency, at least, and often in well-developed cases, it is not obvious, nor, in the main, is it manifest through any objective technic. Indeed, of the potentially lethal disabilities, early coronary insufficiency may be considered the most subjective of all.

Clinical types of coronary insufficiency

Myocardial anoxia in the presence of coronary narrowing may be transient, may be excited by a temporarily excessive demand upon the muscle and may not be associated with structural change in the myocardium. All gradations occur, however, up to the point of myocardial infarction without coronary occlusion, or, finally, to extensive infarction and myomalacia following closure of major coronary vessels.

Clinical responses to this process of ischemia may be classified as (1) asymptomatic degeneration, (2) angina pectoris, (3) congestive heart failure, (4) acute myocardial infarction and (5) disturbances of rhythm and conduction. It is recognized that there exist variations of coronary insufficiency permitting a classification of greater detail in the zone between a short attack of angina pectoris and a prolonged seizure of acute myocardial infarction. The diagnosis of the lesser degrees of injury rests largely upon electrocardiographic evidence of negative displacements of the S-T interval during the attack rather than upon the development of the typical changes of myocardial infarction.

There is no great value in attempting too explicit an analysis of these degrees of ischemia since the prognostic import is never definite—patients die of angina pectoris without coronary occlusion and often survive massive coronary closure. The clinical evaluation is the best guide. Regardless of the electrocardiographic findings, patients, in general, do best who do not develop cardiac enlargement, tachycardia or gallop rhythm following prolonged anginal pain.

Asymptomatic degeneration of the coronary arteries

It may be paradoxical to refer to asymptomatic pathologic changes in the coronary arteries as a clinical response but it is included to point the lesson that high degrees of change may occur in the coronary arteries without symptoms and without producing myocardial ischemia and failure. It is possible to demonstrate calcification of the coronary arteries by roentgenograms during life in individuals free from angina pectoris or congestive signs. In such cases, the coronary arteries are sufficiently capacious to permit satisfactory blood flow to meet the metabolic demands of the particular individual even though they may be rigid and not distensible in the calcified areas. It would seem likely, however, that capillary dilatation is possible distal to these areas with a resulting satisfactory pressure gradient and increase in coronary flow.

Angina pectoris

The interpretation of symptoms to establish the diagnosis of angina pectoris is one of the most important technics in medicine. It is important chiefly in that it places upon the physician the responsibility for applying to a patient the label of an unpredictable and fatal disease. Unlike cancer, angina pectoris is not susceptible to chemical, biopsy or surgical confirmation during life. The diagnosis of angina pectoris can only be mitigated by the skillful avoidance of precise terms in discussion with the patient, and the knowledge that its victims may improve and may live for many years with advice and guidance.

Since the diagnosis rests upon the patient's story, and the doctor's translation, it is difficult for the latter to maintain the critical balance between apprehensive overdiagnosis and neglect of significant atypical symptoms.

Pain.—All physicians and many laymen are familiar with the classical attack of angina pectoris—pain in the chest with extension to the left arm. Such an attack, however, may not be of cardiac but of radicular origin. It is not, in fact, the commonest premonition of anginal distress. Angina is derived from angere—to strangle. The peculiarities of this sensation are that it is a pressure, that it is substernal, that it tends to extend upward, and that it seems to constrict the chest. However, all of these sensations may be mild.

In various patients the sensation may appear as pressure, burning, "raw" feeling, nausea, gas, choking and, less frequently, as pain. No sharp, jabbing, lancinating pain can be considered angina.

The most valuable item in the diagnosis is the substernal position of the distress. The pain, pressure or constriction may originate in the epigastrium, may extend to neck, shoulders, back, arms, wrists, hands, jaw or even the upper part of the abdomen, but at some time in the progress of the attack, in almost all cases, it becomes a discomfort in the middle of the chest. Rarely it remains peripheral, as in patients who have pain in the jaw or the arms on effort. The left arm is often involved but the right arm or both arms not uncommonly. This may be expressed by the patient as a "dead", "weak", or "numb" feeling in the arms.

Precipitants of the attack.—It is a distinguishing feature of angina that it is episodic in nature. It comes in relation to an exciting factor and disappears with its removal. When it does not abate within a few minutes, usually less than three, myocardial anoxia may be presumed to have progressed to a destructive degree, if only at a cytolytic level. The more sensitive the individual, the greater is the likelihood of minor precipitants being effective and the more prolonged the distress after the acute episode has passed. The commonest factors producing attacks are exertion, emotion, eating and exposure to cold. Smoking appears to induce attacks in some.

Such evidence as is available is against use of tobacco in cases of angina pectoris. Coitus often provokes angina and should be inquired about. Nocturnal attacks may be brought on by the recumbent position, presumably by the increased return of venous blood to the heart or by exciting dreams.

The type of exertion responsible for the attack may be relatively specific for a given individual. Walking, especially in the cold, up a mild incline, or after eating, is the most provocative. Use of the arms above the head or even the exertion of shaving will produce distress in some, while others may be able to exercise with their arms without difficulty, yet find walking impossible.

Emotional stress may be a powerful excitant of angina in some people. It need not be of the unpleasant type.

The variability in sensitivity to these precipitants may be striking. Nervous fatigue, worry, excessive use of tobacco or lack of sleep may make the individual much less resistant and, as a result, the attacks may be for a time more frequent or more severe, without necessarily presaging an imminent myocardial infarction or fatal attack.

Other symptoms and signs.—With an attack of angina, pallor often occurs. Dizziness and faintness, even to the point of syncope, occasionally are present. Palpitation is a rare concomitant but is more often the cause of the pain, through the onset of an abnormal tachycardia, than the result of the coronary anoxia. Dyspnea must be differentiated from the pressure sensation of angina although a severe anginal seizure may precipitate an episode of pulmonary edema. Salivation is an unusual but significant sign. Sweating, on the other hand, is almost always present in all but the most evanescent attacks. Belching is common and may accompany the relief of an attack by nitroglycerin. The angor animi, or fear of approaching death, is not often described by the patient in such dramatic terms, but is recognized as an unique sensation so compelling, at times, as to freeze the victim in the path of traffic perils.

Examination of the heart.—It is not the purpose of this paper to describe the abnormalities in the heart which may be found with advanced coronary disease. The more difficult problem consists of the diagnostic criteria of early coronary insufficiency. Unhappily for the diagnosis of angina pectoris, no objective findings exist. In almost one fourth of all patients with this symptom findings on cardiac examination are normal and in the remainder, the existence of cardiac enlargement, murmurs of cardiac dilatation or gallop rhythm are almost always concomitants of myocardial infarction or fibrosis or of an attendant hypertension.

In other words, evidence of heart disease, manifested by physical examination in coronary insufficiency is gratuitous and unexpected. However, there still exists in the minds of many physicians and patients the erroneous belief that by physical examination and electrocardiogram the physician can diagnose coronary disease. When such is possible the disease is far advanced and gross changes in the myocardium are present.

Differential diagnosis.—Attention to details of the patient's history is of the most fundamental importance in the differential diagnosis of angina. It should be the natural desire of the physician to discover some cause, other than coronary disease, for the patient's distress. The commonest of these is the aching precordial pain of nervous fatigue, with or without effort syndrome. Continuous or intermittent, dull or sharp, chest pain which is not precipitated by any distinguishable excitant is not angina pectoris. The causes are to be found in the structures of the chest wall, or pleura, or in the poorly understood inframammary pain of fatigue or apprehension. They are often radicular in distribution. Atypical distress, with emphasis on gastric or epigastric symptoms, should lead to investigation of the gallbladder, duodenum and stomach with a search for peptic ulcer, diaphragmatic hernia or cardiospasm. Even the large bowel, as in diverticulitis, may be incriminated. Cryptic thyrotoxicosis must be kept in mind.

Relief of the attack by nitroglycerin is convincing, but not completely conclusive, evidence of coronary disease, since this drug also relaxes gastrointestinal (especially esophageal) spasm.

Congestive heart failure

Congestive heart failure, as evidenced by dyspnea on exertion, orthopnea, restlessness, insomnia and nocturnal paroxysmal tachypnea with increased venous pressure in pulmonic or peripheral circuits is a commoner response to coronary insufficiency than is angina pectoris. The coronary origin is often established by exclusion of valvular disease, hypertension, thyrotoxicosis, avitaminosis and other less common etiologic factors.

On the positive diagnostic side, the presence of auricular fibrillation, electrocardiographic evidence of conduction defects, or characteristic abnormalities of T waves and S-T segments are strong supports for a coronary anoxic origin of the cardiac failure.

Acute myocardial infarction

An attack of anginal pain, not responding to rest, nitroglycerin or other dilators and prolonged beyond a few minutes may be presumed to signalize a degree of myocardial anoxia to the point of destruction of tissue. Such attacks may occur without actual occlusion of a large coronary artery but usually are the result of the blocking by atheroma or blood clot of a small branch of the coronary arteries. The degree of myocardial necrosis is a function of the size of the occluded artery and the immediately available anastomatic circulation.

An account of the clinical syndrome of acute myocardial infarction is beyond the scope of this paper. Such a condition is recognized readily at the present time. The shock, fall in blood pressure, distant cardiac sounds, fever, friction rub and leukocytosis are accompanied by electrocardiographic evidence of major importance. Following attacks of persistent pain of this sort, serial tracings should always be made since characteristic changes may not appear for as much as one or two weeks after the infarction of a small portion of the myocardium.

Disturbances of rhythm and conduction

Certain abnormalities of rhythm and conduction, only a few of which are susceptible to clinical diagnosis, are strongly suggestive of coronary insufficiency, in the absence of rheumatic valvular disease, acute rheumatic or other infectious carditis, hyperfunction or hypofunction of the thyroid, or pericardial disease.

Of these, auricular fibrillation, auricular flutter and auriculoventricular block with dropped beats can be diagnosed clinically. Auricular fibrillation is very infrequently found with the anginal syndrome. Lesser degrees of auriculoventricular block and intraventricular block are quite common but usually dependent upon electrocardiographic analysis.

Changes in the T waves and S-T segments, unless characteristic of old or recent myocardial infarction, must be interpreted with caution and with due appreciation of the many other factors responsible for such variations.

Stress tests of various types to discover abnormalities in the electrocardiogram, pulse rate or blood pressure when the heart is forced to work under strain will be discussed in other papers in this series.

Summary

The diagnosis of coronary insufficiency of a degree short of myocardial infarction rests, as it did in the days of Heberden, upon analysis of the patient's own story. Physical and roentgenologic examinations are of secondary importance and often valueless. Electrocardiographic study is always indicated but negative findings do not exclude significant coronary narrowing. Coronary insufficiency tends to run its course with exacerbations and remissions largely beyond the influence of our present therapeutic resources. Since the prevention of unnecessary cardiac invalidism, in patients is within the control of the physician, the greatest care should be taken to avoid the diagnosis of coronary disease in unimportant functional disturbances of the heart.

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